CALIFORNIA AGRICULTURAL EXPERIMENT STATION

"ACORN CALVES"

A Nonhereditary Congenital Deformity Due to Maternal Nutritional Deficiency

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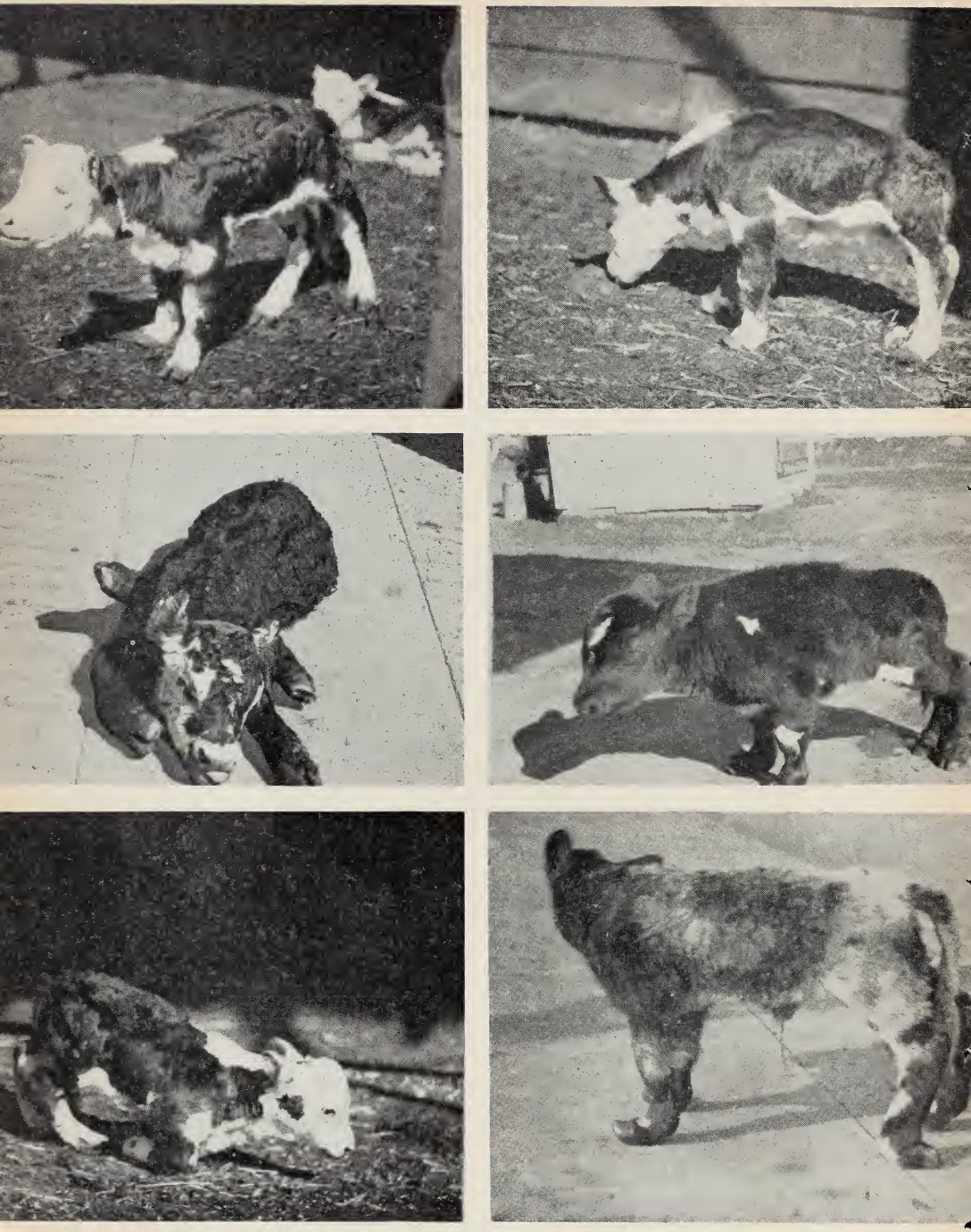

Fig. 1. Typical deformed animals of the type widely referred to in California as "acorn calves."

SUMMARY

not acorns, not inheritance, but maternal nutritional deficiencies cause "ACORN CALVES"

"ACORN CALVES" are more common in the oak belt of the Sierra Nevada foothills than elsewhere. They may be expected in dry years when animals are confined on poor feed in the same areas throughout the year and spend a long time on dry feed. Deformity is more common in calves from heifers in their first pregnancy, but may occur in offspring of cows of any age.

How to Recognize Acorn Calves

Various types of deformity are found in acorn calves, typical examples of which are shown in figure 1 on the facing page. The head may be short, usually with undershot jaw (center, left), or it may be long and narrow (figs. 2 and 9, pp. 8–9 and 18). Usually the long bones of the fore and hind legs are noticeably short (fig. 3). Other abnormalities include incoördination, inability to stand alone, arched back, and a tendency to chronic bloat. This last difficulty is often fatal in animals past the milk-drinking period. Much rarer abnormalities are spasticity in one or more groups of muscles, wry neck, turning in circles, falling over backward, and goose-stepping.

Acorn calves somewhat resemble "bulldog calves," found in Dexter cattle. Bulldog calves, however, are always born dead, usually prematurely, and the deformities are much more extensive than in acorn calves, which are most often born at term and alive. Acorn calves will usually live if helped to nurse during the first week and with good care often reach adult life. Though not economically profitable, they can carry through their normal functions, including reproduction.

What Causes Them?

The bulldog-calf condition is hereditary; the acorn-calf condition is not. Acorn calves have occurred among Hereford, Angus, and Shorthorn breeds. They are not known to have occurred among dairy animals in the irrigated valleys, but they have occurred among dairy animals on dry foothill areas (fig. 10).

Acorn calves are so called because of a rather general impression that they result from the dams' eating too many acorns during gestation. This is not true, but if acorns are the main ingredient of the diet they may prevent the formation or utilization of some essential food element and thus aid in producing acorn calves.

Experiments prove that the condition is due to maternal nutritional deficiencies, probably occurring between the third and sixth month of gestation. Once the alterations in the development of the fetus have taken place, they are not changed by good feed conditions during later months of pregnancy. The specific deficiencies involved have not been found; they are multiple borderland deficiencies, and probably include lack of vitamins A, B complex, and D, as well as protein.

What to Do?

A consistent, constructive policy of livestock management, with supplemental feeding that will enable breeding cows to produce maximum percentage calf crops and calves of optimum weaning weight, can be counted on practically to eliminate acorn calves. (See Extension Circular 131, *California Beef Production.*)

These conclusions are drawn from research and experiments which are discussed in detail on the following pages. The deficient diets which were used in this study appear on pages 13, 14, 15, and 20. The paper which follows represents a progress report on the work on the acorn-calf condition to the present time.

Contents

"ACORN CALVES"

A NONHEREDITARY CONGENITAL DEFORMITY DUE TO MATERNAL NUTRITIONAL DEFICIENCY

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INTRODUCTION

AN ABNORMAL congenital condition studied and described by Hart, Guilbert, and Goss (1932) has existed in cattle on California foothill ranges beyond the memory of the oldest inhabitants. It is not confined to this state, but has occurred in other western range areas. Typical deformities are shown in figure 1.

The anomaly is more common in the oak belt of the Sierra Nevada than elsewhere; and because its incidence has been largely confined to poor feed years, attention has often focused upon the consumption of acorns. Thus the name "acorn calves" for animals so deformed originated because of a rather general impression that the deformities resulted from the dams' eating too many acorns during gestation. Generally, in areas where acorn calves occur, there is a preponderance of the grass species, some filaree, and relatively little bur clover, and the dry feed is inferior. The appearance of the abnormality may be expected in dry years when animals are confined on poor feed in the same areas throughout the year and spend a long period on dry feed. Association with acorn consumption is not consistent; numerous deformed calves have been reported following poor feed conditions with relatively few acorns available, and there have been reports of practically no incidence after a year of heavy acorn mast.

According to reliable accounts, affected calves have been born to cows that did not have access to acorns. Of nine pregnant grade Shorthorn first- and second-calf heifers, kept together in the same dry foothill field in Sacramento County throughout the summer and fall of 1930, five produced deformed calves. One of these is shown in the lower right corner of figure 1. On a small farm in Tehama County, five of the six-cow herd had calves, and all these offspring were deformed. There were no oak trees in either of the enclosures. On the other hand, about 40 out of 700 cows dropped deformed calves in Humboldt County in March and April, 1940. These cows had been on short feed the preceding fall and were said to have consumed many acorns. Deformity is more common in calves from heifers in their first pregnancy, but may occur in offspring of cows of any age.

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REVIEW OF LITERATURE

The anomaly somewhat suggests the recessive achondroplasia-like condition in Dexter cattle which gives affected animals the name "bulldog calves." This has been reported upon at length and definitely associated with heredity by Crew (1923). Bulldog calves are always born dead, usually prematurely; and the anatomical alterations, including umbilical hernia, are much more extensive than in acorn calves. There is excessive fluid in the fetal membranes. This may be recognized as early as the third or fourth month; it obliterates the hollow of the dam's flank. The excess fluid is often discharged through the cervix and vagina, but accumulates again and dribbles until finally the fetus is aborted. The placenta comes away in small fragments. According to herdsmen, there is no afterbirth in these cases, but lochial discharge lasts longer than usual. The death of the fetus if near term is associated with dropsy and difficult parturition. None of these conditions has thus far been reported for acorn calves. Some of the calves we observed and reported on were born dead, but the great majority were born at term and alive. Affected calves will usually live if helped to nurse their dams during the first week or longer.

Wriedt (1930) described a bulldog-calf condition in the Telemark breed of cattle in Norway; the anomaly was traced to one bull, Niklas 481, born in 1899. Although the calves in this account appear more nearly to resemble acorn calves than those reported on by Crew, there is definite evidence that the defect is inherited as a simple recessive. These calves die within a few days.

A new type of recessive achondroplasia in cattle has been described by Gregory, Mead, and Regan (1942). It was uncovered by inbreeding normal dairy animals of the Jersey breed at the University Farm and is inherited as a monofactorial autosomal recessive. It has no effect upon leg length and is usually lethal in both sexes. The greatest modification is in the development of the bones of the skull and jaw—including cleft palate in the more severe cases.

Warkany, Nelson, and Schraffenberger (1943) reported on congenital malformations, particularly cleft palate, induced in rats by maternal nutritional deficiency. They discuss the relation of their findings to the human anomaly. Uebermuth's compilation (1938), cited from different authors, shows that the familial occurrence of the condition in man ranges between 5.7 and 44.5 per cent. More than one affected family member was found in about 20 per cent of the patients studied. No consensus was reached on the mode of inheritance, a fact indicating that the condition is not well understood. The work of Reed and Snell (1931) with harelip in the house mouse was reviewed, and Reed (1936) mentions that these cases occur "when a small number of cumulative genes are present in the homozygous condition provided that chance and environmental conditions are favorable." Stress is laid on the fact that in each of nine pairs of human identical twins, one member showed cleft palate while the other member was normal; this observation proves that nongenetic factors are also involved in the formation of harelip and cleft palate. Warkany, Nelson, and Schraffenberger also refer to the lionesses at the London Zoo which gave birth to cleft-palate cubs on one diet and normal offspring on another. Thirty-two jaguars born to one mother and father in the Berlin Zoo had cleft

palate, but when the diet was changed from cold meat from which the blood had escaped to warm meat containing blood, the same two animals produced 25 more cubs, all of which were normal.

In experiments of Warkany and his co-workers, congenitally malformed offspring resulted when female rats were fed the Steenbock and Black rachitogenic diet No. 2965 (containing 76 per cent yellow cornmeal), supplemented with viosterol. The deformities consisted of shortness of the mandible, shortening or distortion of the extremities, and several other changes, including cleft palate. The experimenters concluded that the malformations resulted from deficiency of the maternal diet, since females of the same strain on an adequate diet did not produce defective offspring. Two per cent of dried pig liver or its alcoholic extract added to the deficient diet resulted in normal offspring even from the same females that had produced deformed offspring on the deficient diet alone. Males siring the abnormal litters from females on the deficient diet, when subsequently mated with females on adequate diet or on the liver-supplemented deficient diet, produced normal offspring. By alternation of the diets producing and preventing the condition, evidence that the factor is stored by the mother was obtained. Finally, by a series of well-controlled experiments with purified maternal diet, in which the various factors of the vitamin-B complex were supplied in crystalline form, Warkany and Schraffenberger (1944) conclusively demonstrated that the deformity was caused by lack of sufficient riboflavin in the mother's diet.

Hale (1934) produced defective offspring in sows deficient in vitamin A during the early stages of pregnancy but later given an ample supply. The defects consisted of lack of eyeballs, accessory ears, cleft palate, harelip, and misplaced kidneys. In the light of the work of Warkany and his co-workers, deficiency of vitamin-B factors may also have been involved in the cases studied by these investigators.

Mellanby (1944), in the Croonian lecture, reviewed his work and that of others since 1918 on nutrition in relation to bone growth and the nervous system. Vitamin A was discovered in 1913. When vitamin D became recognized in 1918 as the curative agent of rickets, there still remained to be studied the syndrome of incoördinated movements previously developed in animals on diets deficient in both vitamins A and D. In 1926 Mellanby began further work on the incoördination and in 1930 he proved the cause to be a deficiency of vitamin A. This condition occurs in several species; Mellanby's work was done with puppies, rabbits, and rats. A variety of symptoms are manifested, depending on the muscles involved; they include loss of vision, hearing, and even sense of position.

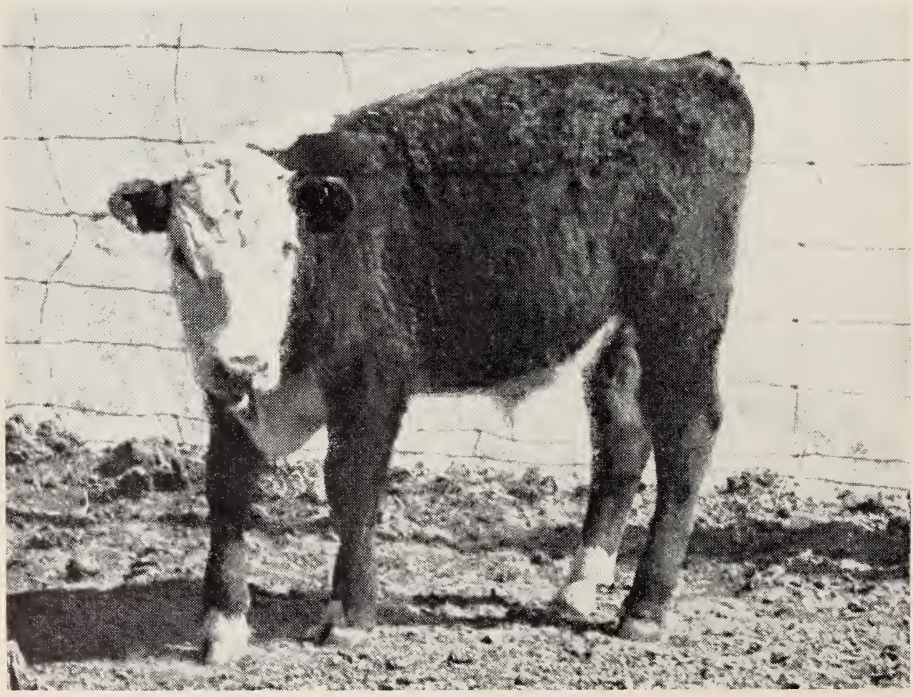
Further investigation showed that the muscular incoördination resulted from lesions of the peripheral nerves and central nervous system in vitamin-A-deficient animals. This condition involved both cranial and spinal nerves. The perplexing problem at the time was that in spinal nerves the posterior or afferent root on one side would be degenerated, while the corresponding root on the other side would be normal or nearly so. In the species studied, the spinal nerves in the cervical region were more degenerated than those of the lumbar, and the dorsal showed least alteration. The cochlear division of the auditory nerve was even more affected than the vestibular division; it had not yet been

observed that the animals were often deaf. In the central nervous system the maximum degeneration was found between the pons and the lower cervical cord.

More years elapsed. Although the muscular incoördination was evidently caused by the nerve degeneration, the latter had not been explained. Further work, started with careful microscopic studies of the internal ear of affected animals, proved that newly formed bone in the modiolus and internal auditory meatus was destroying the auditory nerve. After this finding, extensive investigations showed the bone changes to be widespread in other parts of the body of vitamin-A-deficient experimental animals. These changes proved to be the cause of the nerve degeneration. The nerve pressure and atrophy resulted not only from bone overgrowth but also from absence or diminution of absorption of older-formed bone—a process which is necessary if foramen and other bone cavities are to enlarge with general growth. This work was all done with postuterine experimental animals.

In the problem under consideration here, the congenital blindness of calves is of interest. This condition was first described by Crocker (1919) in newborn Guernsey calves and was termed insidious rachitis. Later, de Schweinitz (1931) and de Schweinitz and De Long (1934) studied what was probably the identical condition in Guernsey calves under the name of papilledema or choked disk and suggested that it might be hereditary.

Moore, Huffman, and Duncan (1935) again described this condition; they regarded it as a type of nutritional blindness, but stated that it apparently differs from true vitamin-A blindness. Congenital blindness was experimen-



tally produced and recorded by Guilbert and Hart (1935) and Hart and Guilbert (1937) in their new-born, vitamin-A-deficient animals; it was considered to be a manifestation of the deficiency. Later work by Moore (1939) and Moore and Sykes (1940) confirmed the relation of the symptoms to vitamin-A deficiency. The cause was shown to be increased cerebrospinal-fluid pressure, resulting in constriction of the optic canals in the sphenoid bone, with consequent optic-nerve atrophy. Increased intracranial pressure also explained incoördination and convulsions in cattle.

Evidently, therefore, nutritional deficiencies can be present in the pregnant female only to the limited extent compatible with a normal gestation period and yet so affect cell differentiation that deformities will exist at birth.

DESCRIPTION OF THE ACORN-CALF CONDITION

The clinical and anatomical variations in acorn calves almost defy description. To judge from scores of cases in many different localities, the condition results from multiple borderland deficiencies, possibly including a lack of vitamin A, B complex, and D, as well as protein. All these essential dietary ingredients, when lacking in the diet of the female during gestation, have been incriminated in abnormal fetal development.

The osseus development of the head in different affected animals is not consistent; there exists the achondroplasia-like condition with shortened head length, usually accompanied by undershot jaw (fig. 1, center), and the long, narrow head development (figs. 2 and 9). Commonly the shafts of the long bones of the fore and hind legs are markedly shortened. (The humerus and the femur from the calf shown in the left center of figure 1 are compared with normal bones in figure 3.)

The alterations may be so grave that the fetus is born dead, or would die quickly if unaided on the range with its dam. Even the calves that can stand

Fig. 2. The steer on the facing page had a long narrow head, a slight arch in the back, a narrow forerib, and a left front leg shorter than the right. Viewed from the side the jawbone appeared straighter than normal, making the muzzle narrow from front to rear. This animal failed to gain normally in the feed lot, became a chronic bloater. He is shown at the right of the lower picture with a similar animal (second from the left) among normal calves of the same age. The photographs were taken at the time of weaning.

nurse with difficulty and frequently require assistance: their short leg bones make it difficult or impossible to reach the teat. Apparently the muscles and tendons develop normally, but the shortness of the bones allows the joints to bend abnormally before receiving muscular support. This condition improves after a few days, and with good care the animals often reach adult life. Though not economically profitable, they can carry through their normal functions, including reproduction.

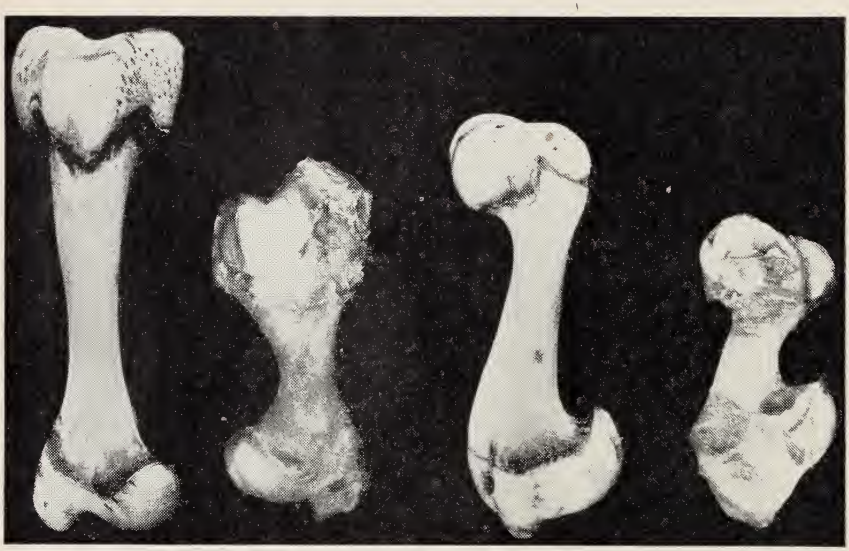


Fig. 3. The leg bones of the deformed calf shown at the left center of figure 1 compared with those of a normal calf to show the short shafts of the former. The two at the left are femurs; the two at the right, humeri.

Other clinical symptoms in acorn calves include incoördination, inability to stand alone, arched back, and a tendency to chronic bloat. This last difficulty is often fatal in animals past the milk-drinking period. It is particularly manifested if the more mildly affected calves reach the feed lot, where with heavy rations they readily go off feed, fail to make appreciable gains, and become a serious financial liability even though death does not occur.

The skeleton of a severely affected calf was carefully boiled out and mounted at the San Joaquin Experimental Range. The bones appeared similar to those of Mellanby's vitamin-A-deficient experimental animals. On the two sides of the median line there was lack of symmetry, particularly noticeable in the foramen magnum in the occipital bone, and the cranial and spinal nerve foramina. The bodies of the lumbar vertebrae were underdeveloped, with arching of the spinal column in this region. Figure 4 shows the skeleton.

The right eyeball of this animal was smaller than the left, with cloudiness of a portion of the right cornea; and the optic nerve on the right side was smaller than on the left. The optic canals in the sphenoid bone, however, were normal; and the nerve on the right side had not been pinched off. In this respect, the condition did not show the characteristic alterations found in calves congenitally blind through vitamin-A deficiency.

The long bones of the legs are first laid down in hyaline cartilage, and this process occurs early in fetal life, through the third or fourth month. Case histories indicate that adverse feed conditions at this time are important in the development of acorn calves. Once the alterations in fetal development have taken place, they are not changed by good feed conditions during subsequent months of pregnancy. The bony changes somewhat resemble those de-

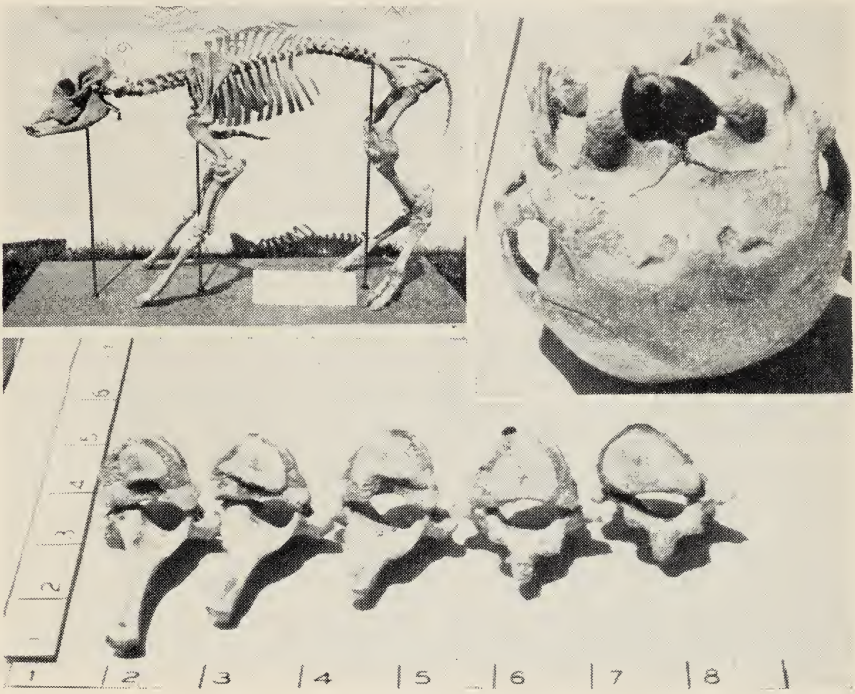


Fig. 4. The mounted skeleton, skull, and individual vertebrae of an acorn calf. The calf was small, but not conspicuously disproportioned compared with the animals shown in figure 1. Practically every bone and articular surface showed abnormalities and malformations. Note particularly the arch in the dorso-lumbar spinal region, the crooked legs due to malformation of articulations, the distortion of the left (right in photo) occipital condyle on the skull. The bodies of most of the vertebrae were lopsided, and the foramina for the spinal cord were flattened or otherwise distorted from the normal round formation, as is shown in the lower part of the figure.

scribed in the literature under the terms *chondrodystrophy* and *mikromelia*. Affected calves do not evidence much, if any, disorderly proliferation of cartilage cells, or periosteal bone formation; the epiphyses do not swell, but rather there is a failure of normal growth. The bony tubercles for the muscular insertions develop prominently. Dentition is normal. Although changes in the ribs and sternum are not marked, definite swelling pseudorosary sometimes occurs at the junction of the ribs and their cartilages.

Much rarer abnormalities include spasticity in one or more groups of muscles, wry neck, turning in circles, falling over backward, and goose-stepping. In one wry-necked calf there was ankilosis of the occipitoatloid articulation, so that the head had to be chopped off in the packing-house; the usual disarticulation with a knife was impossible.

EXPERIMENTAL DATA

Normal Offspring Produced from Affected Sires and Dams.—To prove whether or not the acorn-calf condition was hereditary, a group of affected calves of both sexes was assembled at the San Joaquin Experimental Range, beginning in 1937, so offspring might, if possible, be produced from affected sires and dams. In all, nine animals were secured from herds near the Experimental Range. One bull and two heifer calves were brought to the University Farm to be raised until past the milk-feeding stage. One of the heifers died of bloat at the Farm, and the other died from the same cause after being weaned and sent back to the Range. Figure 5 shows the bull shortly after birth, again at 2 months, and finally at 7 months. The badly deformed heifer also shown, about 7 months old, is the one that died when returned to the Range. Though abnormally shaped, the bull became vigorous; he sired three offspring from two affected females. One other male and four females were acquired as weaners. Of these, the bull and two heifers died of bloat shortly after arrival at the Range, probably because the change from milk to all dry feed had accentuated the tendency to bloat. The other two heifers were accidentally bred when normal bulls at the Range Experiment Station broke into their pasture; both gave birth to normal calves. One heifer suffered eversion of the uterus and was therefore destroyed. The other animal was later hand bred to the deformed bull. The resulting heifer calf was entirely normal and produced two normal offspring sired by normal bulls. When the deformed dam was again bred to the deformed bull, the result was a normal male with a peculiarly curled coat. It was castrated, raised, and sold as a two-year-old feeder steer. The dam, now having produced three calves, came into estrum and was bred; but she failed to conceive, gradually became emaciated, and died. The bull and the cow at maturity, with their two normal offspring, are shown in figure 6. All these animals were predominantly of Hereford breeding. One Aberdeen

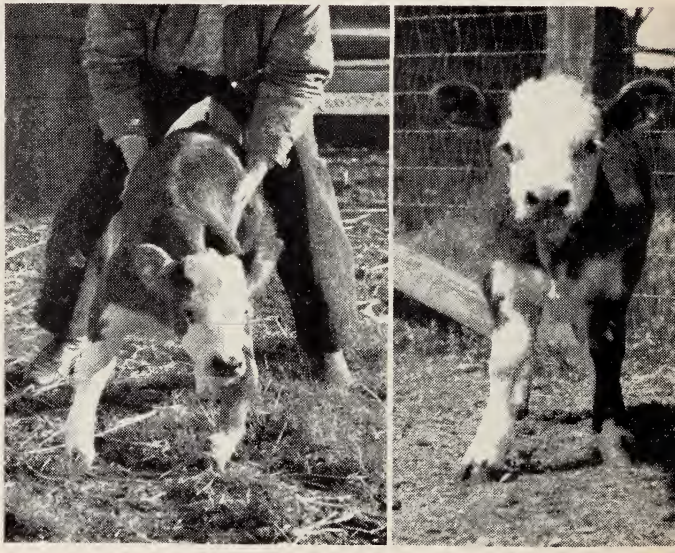


Fig. 5. From left to right are three photographs of the deformed bull used in the breeding tests, taken at a few days, 2 months, and 7 months, respectively. The badly deformed heifer shown in the far right was also raised by hand, but died of bloat when she was returned from Davis to the San Joaquin Experimental Range. The bull's legs straightened within a couple of weeks after birth. Note the shortness of legs compared with body length at 7 months, and also the slight arch in the back.

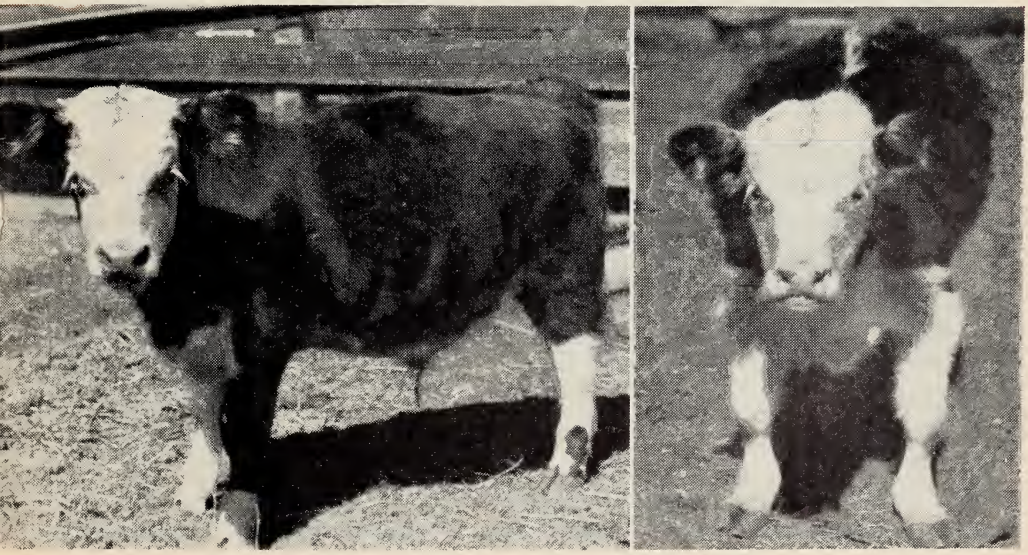
Angus female (fig. 7), markedly deformed, was purchased when three years old and was bred for the first time to the deformed bull. She conceived but at the end of gestation died from the mechanical difficulties resulting from the development of a normal-sized male fetus in her badly deformed body. A post-mortem examination showed the fetus to be normal.

Observations were made on a neighboring ranch where a deformed Hereford bull (fig. 8) had been bred to normal cows in a dairy herd. He had sired all normal calves; ten of the 1936 calves were actually seen and examined.

Most defects are recessive; the affected animals have received a gene for the condition from both parents. Such animals necessarily breed true, and if bred together always produce defective offspring. Thus one normal offspring from defective parents would prove the abnormality was not caused by a recessive hereditary factor. A dominant character, on the other hand, can be inherited even from one parent; then, on the average, half the offspring from the abnormal animal are affected. The data presented above eliminate the possibility of either dominant or recessive causative factors and prove conclusively that the condition is not hereditary.

Since the acorn-calf deformity has proved to be environmental in origin, attempts have been made to identify the nutritional deficiency involved.

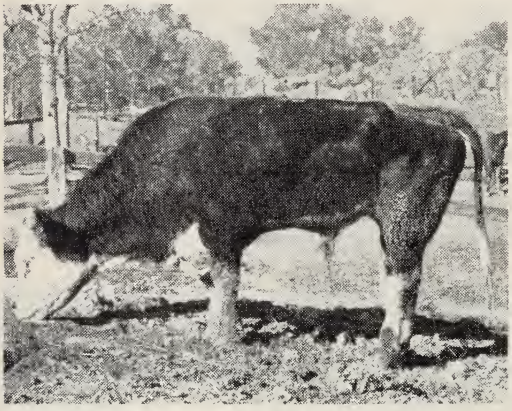
Acorn Feeding Experiments.—In 1936 three four-year-old females, numbers 5, 52, and 65, which had previously failed to conceive and were then pregnant with their first calves to normal bulls at the Range, were fed acorns in addition to the natural range forage. By August 5, when the experiment started, they were respectively in the 28th, 24th, and 14th weeks of pregnancy. The acorns were from the blue oak, *Quercus douglassii*, the common deciduous oak of this area. The animals lost 167, 169, and 107 pounds respectively between August 5 and October 1, 1936, and an average of 3.13 pounds daily during September. Numbers 5 and 65 gave birth to normal calves. Number 52, which consumed about 1,300 pounds of acorns during the last 122 days

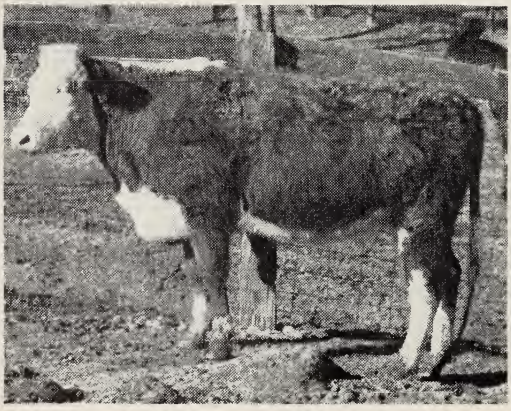


of pregnancy, gave birth to a 31-pound deformed calf. This offspring (calf 13) is shown in figure 9 in comparison with a normal calf of the same age, 30 days. Its crooked spine and a hump behind its shoulder became worse with time; and at about two and a half years (shortly after the picture on the right in figure 9 was taken) the animal died from bloat. The long, narrow head resembles those of animals shown in figure 2, but these were not otherwise so conspicuously deformed.

One typical acorn calf, a male, was born on the range in the winter of 1941–42 to a cow that was fed no supplementary feeds; the calf was sired by a normal bull. Very few acorns were available that season. A stillborn animal with crooked legs, born in the supplemented herd, was also considered an acorn calf. One severely spastic calf and at least two calves having only wry neck were born. Several slow-growing calves with peculiar long narrow heads and a tendency to bloat were produced in a supplementally fed herd that was

Fig. 6. The bull, top left, which is shown as a calf in figure 5, when mated to the cow on the right produced the two normal offspring shown below. The cow, more severely deformed as a calf, had the characteristic arch in the back, and her right foreleg was crooked. The extreme length of the bull compared with his height is somewhat accentuated because of thinness after a pelvic injury. Both calves, when a year old, were taller than the mother. Note especially the long legs of the steer, lower right.





confined year-long to the same pasture on the Experimental Range. Except for a single animal with one front leg shortened, these calves were not conspicuously disproportioned in their bodies. (See figure 2.)

Failure to Develop Acorn Calves on Low-Protein Rations.—Acorns are low in protein, and their tannin content may intensify the protein deficiency of the range feed by rendering the amount present less available. In experiments, small amounts of cottonseed cake, fed to supplement acorns and dry range feed, enabled the animals to gain weight. These findings strengthened

Fig. 7. A markedly deformed three-year-old cow of Aberdeen Angus breeding. The bulldog-type head was particularly conspicuous in this animal. Bred to the deformed bull shown in figures 5 and 6, she produced a calf which was normal at term, but which could not be delivered and therefore caused the death of the mother.

the postulation that protein starvation at a critical stage of pregnancy—a stage placed, by many case histories, between the third and the sixth month of gestation—might be involved in the deformity.

In November, 1937, six pregnant heifers from the University herd, varying in age from 16 to 21 months, were placed on a low-protein ration: well-bleached barley straw, 76 per cent; cane molasses, 24 per cent. This mixture and salt were fed *ad libitum* with 0.1 pound of calcium carbonate daily. Its percentage composition was as follows:

	Per cent
Moisture	9.53
Crude protein	2.13
Ash (including 0.39 per cent calcium, 0.05 per cent phosphorus, and 4.14 per cent SiO_2)	8.81
Crude fiber	29.53
Fat and nitrogen-free extract	50.00
Total	100.00

The ration is very low in crude protein and phosphorus, but not beyond the limit of dry range feed in poor years.

Some of the data on the animals are given in table 1.

The heifers were placed on the rations November 18, 1937; and on January 26, 1938, all were found (on rectal palpation) to be pregnant.

The animals on the basal ration rapidly lost appetite and consumed only 4 to 8.6 pounds daily; but the two controls, each given an additional 2 pounds of cottonseed meal daily, consumed an average of 12 and 15 pounds of the straw-molasses mix. Early in the experiment the animals on the basal ration were found to be obtaining, through the fence, some feces from horses kept in an adjoining lot. This was thereafter prevented by tight boarding of the corral fence on that side.

To test whether there was a difference among the experimental animals in craving for abnormal feed, tankage was once offered to the heifers. Of the animals on the basal ration, numbers 579 and 588 ate it avidly, while the other two showed some interest but did not eat. Numbers 584 and 585, receiving cottonseed, were not interested in tankage.

Extraction of feces collected January 17 from two animals on basal rations and from number 585 showed no carotene or only a trace.

Since the heifers had been continuously on green feed, with ample opportunity for storage before they were placed on experiment, no vitamin A was supplied at first. During the last 2 months of gestation, the animals receiving cottonseed and two of the four animals on basal ration (numbers 584, 585, 000, and 588) received cod-liver oil. At no time did they show symptons of vitamin-A deficiency.

Heifers 584 and 585 were left on the same rations until calving. Number 589 was moved to green pasture April 27, and number 579 on May 21, to simulate the conditions under which most deformed calves are produced. The other two animals, 000 and 588, were continued on the basal ration until the beginning of the eighth month of gestation and then 2 pounds apiece of cottonseed meal with cod-liver oil was added to their daily ration.

The control animals, 584 and 585, maintained good condition and produced thrifty calves weighing 56 and 76 pounds respectively. The other four became extremely thin, but gave birth to normal small calves weighing 48 to 51 pounds. The cows were slow in getting back to normal. This very low protein intake with loss in weight is evidence that the pregnant animal can, within limits,

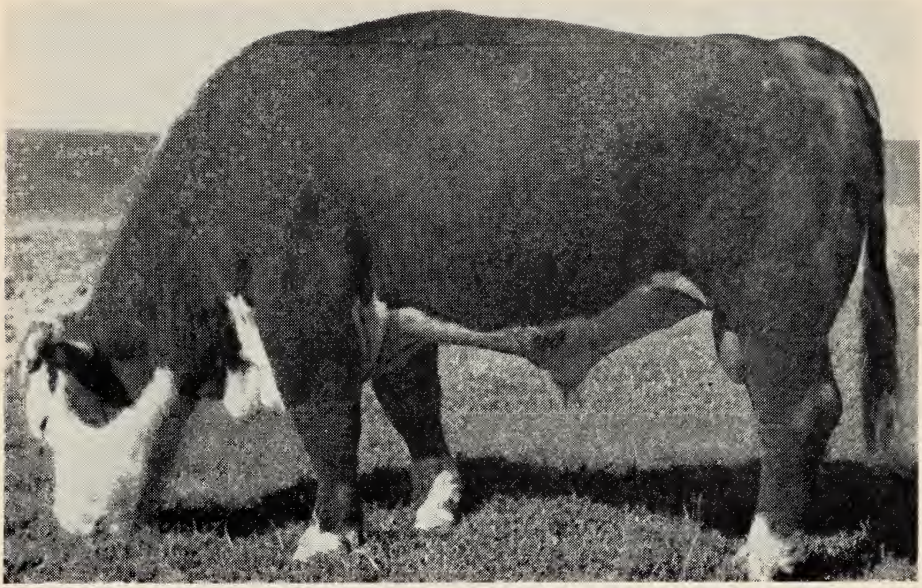


Fig. 8. Left and above, a small bull that was deformed as a calf and at maturity had an arched back and a peculiarly shaped head. He was bred to about 20 cows in a dairy herd, and all offspring were normal.

draw from her own body necessary protein to develop a normal fetus. The presence of molasses in the diet would promote bacterial growth in the rumen, thereby favoring synthesis of the factors in the vitamin-B complex that may be lacking on dry range feed alone. Molasses also contains appreciable thiamin, riboflavin, and pyrodoxin, with larger amounts of niacin and pantothenic acid.

The experiment showed strikingly how cottonseed-meal supplement increases appetite and feed consumption. It showed also that successful reproduction was possible on a ration restricted to straw, molasses, salt, calcium carbonate, and cod-liver oil, with or without cottonseed-meal supplement. Though the calves born to these cows were small, there was no evidence of deformity. The results of this experiment and other observations have shown no conspicuous drop of blood phosphate on very low phosphorus rations when protein and total feed intake were low and weight losses were rapid.

Low-Manganese Rations.—An experiment was carried out in an attempt to obtain low manganese intake during pregnancy.

Manganese is one of the trace elements required in nutrition, and its deficiency in intrauterine life has been shown to affect growth so that bones will be shortened and thickened, the ash content modified, and strength of the union between epiphysis and diaphysis reduced. In most of these experiments chickens and turkeys were used, but similar results have been obtained with rats.

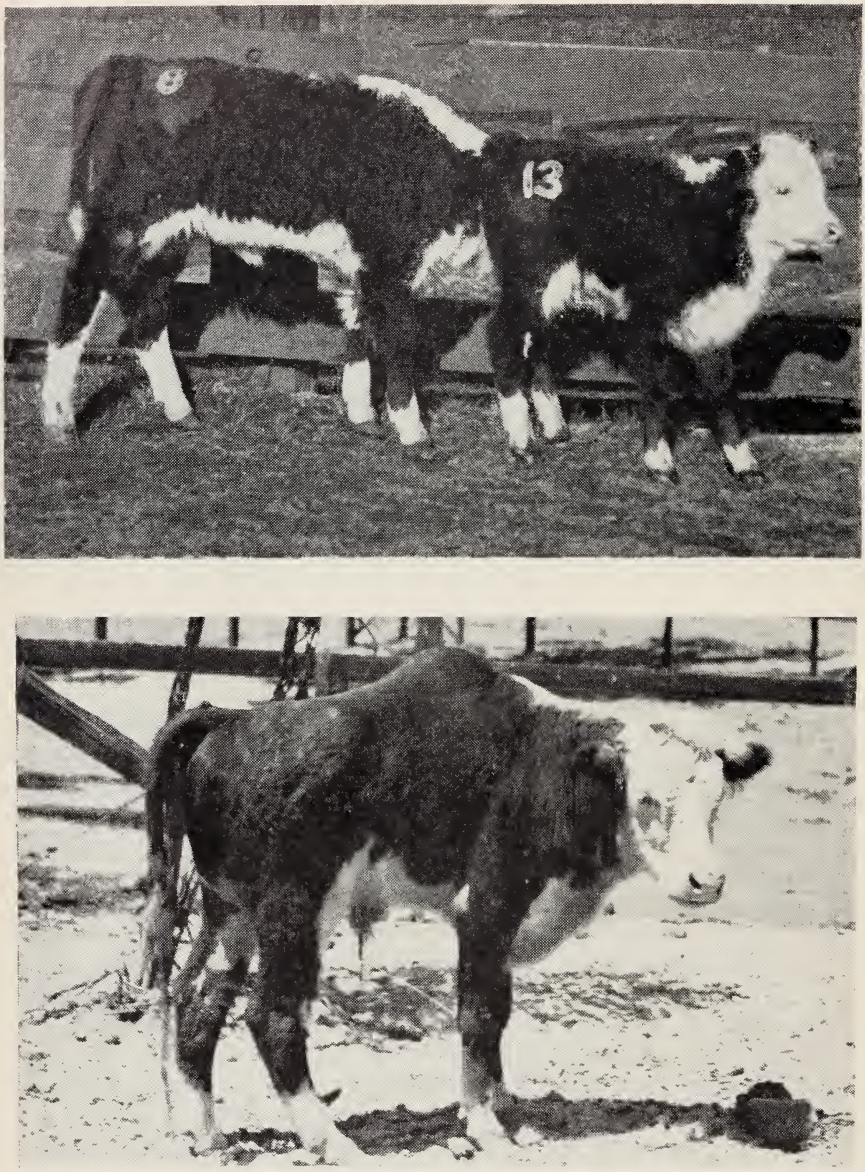


Fig. 9. The small calf, No. 13, from cow 52 which received acorns from the twenty-fourth week of pregnancy to parturition, is shown in top photograph with a normal calf of the same age (30 days). Its back was somewhat humped at birth and became progressively worse, as is shown in the lower picture, taken at 2½ years, when he weighed about 400 pounds. The long narrow head is similar to those of the animals shown in figure 2, which, however, have no other conspicuous deformities. Photo by H. H. Biswell, California Forest and Range Experiment Station.

Eight Hereford heifers from the San Joaquin Experimental Range were brought to the University Farm for the experiment. The ration on which they were placed had the following ingredients:

	Per cent
Yellow cornmeal	60.0
Commercial casein	10.0
Ground wood sulfite paper pulp	15.0
Corn sugar (cerelose)	10.5
Bone meal	2.0
Precipitated calcium carbonate	1.0
Salt	1.0
Mineral mixture	0.5
Total	100.0

The mineral mixture was added to insure adequate amounts of trace elements except manganese and was made up as follows:

	Per cent
Ferric citrate	17.0
Calcium sulfate	2.1
Zinc sulfate	1.0
Magnesium carbonate	22.0
Cobalt carbonate	0.1
Potassium iodide	0.3
Potassium chloride	57.5
Total	100.0

Analyses were run for the manganese content of the mixed basal ration; it contained 6.6 parts per million of manganese.

The heifers were divided into three lots: group 1, four animals, had a limited feed intake of 8 pounds per head daily of the basal ration; group 2, two animals, fed more liberally of the same ration, averaged 11.9 pounds daily during the 305-day period; group 3, two control animals, were given the limited 8 pounds per day of the basal ration, to which manganese carbonate had been added to bring the manganese content of the ration to 200 parts per million.

Shark-liver oil of high vitamin-A potency (100,000 U.S.P. units per gram) was fed at irregular intervals so that the intake averaged about 40,000 units daily per animal.

The heifers were maintained in two concrete-floored lots partly covered by a shed roof. At each feeding they were placed in individual feeding pens and fed from concrete mangers. The inclusion of paper pulp did not satisfy their craving for roughage; they chewed the corral fence till it had to be covered with woven wire.

The first few weeks on the ration, the animals scoured, but this trouble subsided and thereafter occurred only sporadically. The digestibility of the ration was high. The feces were scant, abnormal in appearance for ruminants, and objectionable in odor. No bloating was observed at first, but after about 3 months all animals tended to show considerable distention of the rumen shortly after eating. Bloating was a limiting factor in the amount that could be fed group 2. This observation suggests that rumen activity and belching decline after a period on a ration lacking rough, stimulating material.

TABLE 1

DATA ON PREGNANT HEIFERS IN LOW-PROTEIN EXPERIMENT

Heifer no., breed, and date of birth	Stage of pregnancy, Nov. 17, 1937	Initial weight, Nov. 17, 1936	Final weighing date, 1938	Final weight	Gain or loss	Serum phosphorus		Serum protein		Serum carotene, Feb. 26, 1938	Calving date, 1938
						Feb. 18, 1938	Mar. 24, 1938	Feb. 18, 1938	Mar. 24, 1938		
	months	*pounds*		*pounds*	*pounds*	*mg per 100 cc*	*mg per 100 cc*	*per cent*	*per cent*	*mg per 100 cc*	
Heifers on basal ration:											
No. 000, Hereford × Shorthorn cross, born May 16, 1936	2½	890	June 14	690	−200	6.1	4.9	6.3	6.5	None	June 12
No. 579, Angus, born Jan. 11, 1936	1	1,000	May 25	750	−250	6.3	6.6	6.8	6.6	None	July 22
No. 588, Hereford × Shorthorn cross, born Mar. 18, 1936	1	915	July 14	730	−185	6.0	6.8	5.8	5.9	None	July 21
No. 589, Shorthorn, born May 19, 1936	2	915	June 18	700	−215	4.7	6.3	6.3	6.1	None	June 20
Heifers on basal ration + 2 pounds daily of 43 per cent cottonseed meal:											
No. 584, Angus, born Feb. 25, 1936	Bred this date	945	June 15	993	+38	6.2	6.6	6.9	7.0	0.12	Aug. 24
No. 585, Hereford × Shorthorn cross, born Feb. 26, 1936	2	1,010	July 2	1,005	−5	6.9	5.9	6.8	7.0	None	June 21

A bull was maintained in an adjoining lot, and the heifers were bred at the first estrum after going on the experiment. Four became pregnant at the first service, two required a second service, and the remaining two required three services. The cows had been placed on the rations February 15, 1941; the last one calved April 4, 1942. All offspring were normal.

Phosphatase determinations were made on blood-plasma samples drawn on four occasions between September 24, 1941, and January 31, 1942, from all the animals. There were 4.4 to 10.1 phosphatase units per 100 cc of blood plasma in groups 1 and 2, on the low-manganese ration, and 6.4 to 11.0 units in group 3; but the differences were not significant. At the time of the experiment it was ascertained that soil near the concrete-floored enclosures contained 560 parts per million of manganese. Thus extra intake of manganese from blown dust was not under control, although the pavement was frequently washed off. The experimental animals were probably not much reduced in the very minute quantities of manganese required. This diet proved adequate for gestation and lactation. The principal defect apparent was lack of fiber with the physical properties essential to the stimulation of normal rumen activity.

The ration was highly concentrated, digestible, and efficiently utilized. Group 2, which averaged 480 pounds in weight at the start, gained 2.03 pounds daily during the first 58 days, consumed 8.6 pounds of ration daily, and made 100 pounds' gain on 425 pounds of feed (a figure comparable with that for swine). During the next 56- and 59-day periods respectively, this group gained 1.79 and 1.95 pounds daily, consumed 11.1 and 12.6 pounds of feed, and required 622 and 647 pounds of feed for 100 pounds' gain. Because the feed was not increased in relation to the progressively greater size of the animals, the gains became smaller and efficiency went down as the experiment progressed. Over the entire period the average daily gain was 1.62 pounds, and 734 pounds of feed was required per 100 pounds' gain. The groups on more limited feed gained about 1 pound daily and required 740 and 864 pounds of feed respectively for 100 pounds of gain.

DISCUSSION

From the beginning of our observations on acorn calves there was no evidence that the condition was hereditary. The origins of the affected animals varied greatly. They included all three beef breeds—Hereford, Angus, and Shorthorn—that make up the great bulk of range cattle in this country. Two normal calves were produced from one deformed grade Hereford cow, and one normal fetus was found at term from a markedly deformed grade Aberdeen Angus female; all three had been sired by one deformed grade Hereford bull. All the achondroplasia conditions found in cattle have been recessive defects and therefore must be homozygous for phenotype manifestation. Since the mating of defective sires and dams produced normal offspring, the acorn-calf condition is eliminated as a recessive as well as a dominant hereditary defect. Having demonstrated that this abnormality was not hereditary, we have tested various theories regarding its possible etiology. We have made some unsuccessful attempts to produce the condition.

Now that vitamin-A deficiency is becoming better understood, it is evident that this factor cannot be entirely eliminated in a study of acorn calves. Vita-

min-A deficiency resulting in stillbirths and abortions usually occurs when the dry season is prolonged to November or December, particularly among bred heifers and cows approaching term at this time. Acorn calves, on the other hand, are more commonly born between February and June, after green feed has been available for some time. The defects in swine reported by Hale (1934) were produced by conditions present only at certain stages of pregnancy.

Fig. 10. A deformed calf from a dairy cow developed under foothill-range conditions.

The skeleton of one affected animal on examination did show variation in size of foramina on one side of the median line as compared with the other side and irregularities in the symmetry of the spinal canal, as observed by Mellanby in his experiments with animals of other species on diets low in vitamin A.

In poor feed years vitamin A is deficient over a long period on rangelands. Acorn calves have never occurred among dairy animals in the irrigated valleys, but they have occurred among dairy animals on dry foothill areas; such a calf is shown in figure 10.

Congenital blindness due to constriction of the optic nerves in the optic canals of the sphenoid bone, although definitely known to result, in calves, from maternal vitamin-A deficiency, has never been demonstrated in acorn calves. The shortness of the long bones of the legs has no definite explanation at present. Under the most severe feed conditions on ranges the incidence of deformity reaches only about 15 per cent of the calves born, except in occasional situations on small enclosures with few animals.

With the development of knowledge concerning the synthesis of various factors of the vitamin-B complex by microörganisms in the rumen of cattle, there was a tendency to eliminate those factors as possibilities in the etiology of the acorn calf. Further work, however, has demonstrated that the type of organisms and of fermentation, and the amount of various B-complex components synthesized, vary with the constituents of the ration that forms the substrate in the rumen. In a preliminary examination, the rumen contents of cattle subsisting on nutritionally deficient dry range forage have appeared to be low in riboflavin. In view of the findings of Warkany and his co-workers, this observation may be significant. Certainly there remains the possibility that B-complex factors contribute to some of the manifestations described. In the low-protein experiment, molasses was added to the ration. This could have improved microbiological synthesis, as well as actually contributing some of the vitamin-B complex.

CONCLUSION

Data on acorn calves have been accumulated over the last fifteen years. It can now be stated that the condition is nonhereditary; that it is due to the maternal nutritional deficiency, probably occurring between the third and sixth month of gestation. The specific deficiency or deficiencies involved have not been ascertained.

Since acorn calves have occurred without the dams' having access to acorns, it is evident that their ingestion is not the direct causative agent. Acorns may be a contributing factor, however, when they are the main ingredient of the diet, by preventing the formation or utilization of some dietary essential.

A consistent, constructive policy of livestock management, with supplemental feeding that will enable the breeding cows to produce maximum percentage calf crops and calves of optimum weaning weight, can be counted on practically to eliminate acorn calves.

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